

**Conclusion:** We suggest that coronary artery remodeling is a major contributing factor to angiographic misinterpretation of disease eccentricity that can be determined accurately only by the IVUS.

### 1217-82 Comparison of the Restenosis Mechanism of Cutting Balloon Angioplasty and Plain Old Balloon Angioplasty: A Serial Intravascular Ultrasound Study

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**Background:** The Cutting Balloon (CB) is a novel dilation device that longitudinally incises the coronary plaque during balloon dilation. This incision limits the degree of arterial trauma associated with balloon dilation, potentially reducing the late injury response compared with plain old balloon angioplasty (POBA).

**Methods:** To evaluate the restenosis mechanism of CB in comparison with POBA, we performed intravascular ultrasound (IVUS) in 148 lesions (98 CB and 48 POBA) before and after the procedure and at a mean follow-up of 6 months. There were no significant differences in the patient and lesion characteristics between the two groups. Vessel area (VA), lumen area (LA) and plaque area (PA) were measured.

**Results:** There were no differences among CB and POBA in pre and post changes in VA, LA and PA.

		CB (n = 98)	POBA (n = 48)	P value
Restenosis	AVA	1.4 ± 2.4	1.4 ± 2.3	NS
	APA	1.5 ± 2.3	1.0 ± 2.4	NS
	ALA	2.0 ± 1.2	2.4 ± 1.7	NS
Non-restenosis	AVA	0.2 ± 2.0	1.2 ± 3.6	NS
	APA	0.4 ± 2.3	1.6 ± 3.2	0.04
	ALA	0.2 ± 2.6	0.4 ± 2.2	NS
Binary restenosis (QCA)		27%	38%	NS

(Δ, changes between post and follow-up)

**Conclusion:** In the restenosis group, there were no differences between CB and POBA. In the non-restenosis group, CB resulted in less plaque increase and plaque increase after POBA caused compensatory enlargement of coronary arteries, which may suggest a smaller degree of arterial trauma by CB.

### 1218 Newer Agents of Vascular Injury

Wednesday, April 1, 1998, 3:00 p.m.-5:00 p.m.  
Georgia World Congress Center, West Exhibit Hall Level  
Presentation Hour: 3:00 p.m.-4:00 p.m.

### 1218-6 Acute Phase Proteins vs Traditional Risk Factors as Markers of Arterial Wall Impairment

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**Background:** Acute phase proteins (APP), such as C reactive protein (CRP), fibrinogen (FBG) and the 3rd complement component (C3), are associated with the risk of ischemic events, often more closely than traditional risk factors (TRF). In this study the relation of APP and TRF with arterial wall impairment has been assessed.

**Methods:** In 317 unselected men aged 55-64 years, carotid and femoral arteries were examined at the bifurcation by ultrasonography. For each subject the maximum intima-media thickness (IMT) and the maximum % stenotic area were related to CRP, FBG, C3, C4 and TRF.

**Results:** IMT correlated with CRP (Spearman's  $\rho = 0.21$ ;  $P = 0.0004$ ), FBG ( $\rho = 0.19$ ,  $P = 0.0013$ ) and C3 ( $\rho = 0.12$ ;  $P = 0.0374$ ). Stronger correlations were found, however, with TRF such as cigarettes/day ( $\rho = 0.38$ ;  $P = 0.0001$ ) and cholesterol ( $\rho = 0.26$ ;  $P = 0.0001$ ). Twenty-one subjects had at least one arterial stenosis  $\geq 50\%$ ; compared with the rest of the sample, they had higher levels (mg/dl) of C3 ( $80.6 \pm 12.5$  (1 SD) vs  $73.8 \pm 11.3$ ;  $P = 0.0274$ ), C4 ( $33.8 \pm 8.5$  vs  $29.7 \pm 7.2$ ;  $P = 0.0380$ ), CRP ( $0.38 \pm 0.33$  vs  $0.29 \pm 0.41$ ;  $P = 0.0669$ ) and FBG ( $277.6 \pm 67.6$  vs  $252.6 \pm 60.8$ ;  $P = 0.0784$ ). In multiple logistic regression, of these 4 APP only C3 was independently associated with a stenotic area  $\geq 50\%$  ( $P = 0.0165$ ) but, after addition of TRF, only triglycerides ( $P = 0.0008$ ), systolic blood pressure ( $P = 0.0111$ ) and cigarette smoking ( $P = 0.0164$ ) remained in the model, and all APP were excluded.

**Conclusions:** The quantitative relation of arterial wall impairment with APP is weaker than the one with TRF. Thus, this relation does not account for the closer association of APP with the risk of acute ischemic events.

### 1218-7 Hyperhomocysteinemia Is an Independent Risk Factor for Carotid Atherosclerosis

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Recent studies suggest a relationship between vascular occlusive disease and hyperhomocysteinemia, which may damage cells lining blood vessels and favor atherosclerosis. This study evaluated if patients with carotid atherosclerosis (CAS) had higher blood levels of homocysteine. CAS was defined as intimal media thickness of  $\geq 2$  mm on the far wall at carotid bifurcation or at internal carotid measured by B-mode ultrasonography. Total homocysteine levels (tHcy,  $\mu\text{mol/L}$ ) were determined by HPLC with fluorescent detector. The study studied 191 consecutive patients (48% males, mean age  $58 \pm 14$  years, 50% dyslipidemia, 41% hypertension, 28% smokers, 14% coronary artery disease, 10% diabetes). Patients with CAS ( $n = 91$ ), when compared to patients without CAS (controls,  $n = 100$ ), had older age ( $64 \pm 10$  vs.  $52 \pm 10$  yrs,  $p < 0.001$ ), male gender (61% vs. 36%,  $p > 0.01$ ) and hypertension (57% vs. 27%,  $p < 0.001$ ). Dyslipidemia, smoke, diabetes, body mass index, serum creatinine, folic acid and vitamin B12 were similar in the two groups. In contrast, tHcy levels were significantly higher in patients with CAS ( $11.7 \pm 6.5 \mu\text{mol/L}$ , 95% confidence intervals (95% CI) 10.3-13.0) than in controls ( $8.1 \pm 4.4 \mu\text{mol/L}$ , 95% CI 7.2-8.9, ( $p < 0.0001$ ). Females had lower tHcy levels than males both in patients with CAS and in controls. tHcy levels were positively correlated with age ( $p = 0.02$ ) and negatively correlated with folic acid ( $p < 0.001$ ), but were not correlated with B12 or creatinine. By logistic regression, independent predictors of CAS were male gender (odds ratio (OR) 2.56; 95% CI 1.30-5.40;  $p < 0.0001$ ), hypertension (OR 2.55; 95% CI 1.28-5.17;  $p < 0.0001$ ), age (OR per 10 year increments 2.15; 95% CI 1.57-2.94;  $p < 0.01$ ) and tHcy levels (OR per unit increments 1.10; 95% CI 1.03-1.19;  $p < 0.005$ ). In conclusion, hyperhomocysteinemia is an independent risk factor for carotid atherosclerosis. Since hyperhomocysteinemia may be corrected by folic acid and vitamin B12 supplements, homocysteine blood levels should be screened in patients at risk of atherosclerosis and vascular occlusive disease.

### 1218-8 Increased Soluble Form of Vascular Cell Adhesion Molecule-1 and Interleukin Adhesion Molecule-1 in Intermittent Claudication

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**Background:** A rapid increase in leukocyte adhesion to endothelium is one of the earliest events in response to inflammation and in the pathogenesis of vascular damage. Vascular cell adhesion molecule-1 (VCAM-1) and interleukin adhesion molecule-1 (ICAM-1) are members of the immunoglobulin gene superfamily that play a major role in the leukocyte adhesion process. We assessed the effect of treadmill exercise on circulating soluble form of VCAM-1 and ICAM-1 in patients with intermittent claudication.

**Methods:** In 12 claudicants and 8 age-matched control subjects, venous plasma levels of VCAM-1 and ICAM-1 were determined by sensitive ELISA tests at rest, at maximal tolerated exercise and 5, 15 and 30 min after exercise. Patients were selected who did not present any condition known to interfere with adhesion molecule modulation.

**Results:** In controls, exercise did not affect plasma levels of adhesion molecules. VCAM-1 was  $610 \pm 42 \text{ ng/ml}$  at rest and  $592 \pm 53 \text{ ng/ml}$  at peak exercise. The corresponding values for ICAM-1 were  $232 \pm 17$  and  $233 \pm 21 \text{ ng/ml}$ . Conversely, all claudicants showed increased plasma levels of both VCAM-1 and ICAM-1 at maximal tolerated exercise. VCAM-1 increased from  $691 \pm 65$  to  $832 \pm 83 \text{ ng/ml}$  ( $p < 0.05$ ). ICAM-1 returned from  $249 \pm 20$  to  $275 \pm 22 \text{ ng/ml}$  ( $p < 0.01$ ). Thirty minutes after exercise, VCAM-1 and ICAM-1 returned to resting values, being  $709 \pm 70$  and  $244 \pm 21 \text{ ng/ml}$ , respectively. No relationship was found between plasma values of adhesion molecules and treadmill performance.

**Conclusion:** In patient with intermittent claudication, exercise increase plasma levels of VCAM-1 and ICAM-1. This probably reflects endothelial activation or damage and thus may have potential implications on the pathophysiology of the arterial disease.

### 1218-9 Inhibition of Arterial Thrombosis: Synergistic Interaction Between Platelet IIb/IIIa Receptor and Tissue Factor

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**Background:** Recent studies have suggested the importance of platelet IIb/IIIa receptor in arterial thrombosis. Equally the release of tissue factor (TF) and its interaction with factor VII, at sites of vascular injury may promote thrombosis.